U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 19 March 2007Case Nos.: 2004-BLA-6645

2004-BLA-6646

In the Matter of:

M. M., Survivor of
B. D. M., Deceased,
Claimant

v.

Omar Mining Company.
Employer

And

Director, Office of Workers' Compensation Programs,
Party-In-Interest

DECISION AND ORDER GRANTING BENEFITS IN LIVING MINER'S AND SURVIVOR'S CLAIMS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

A formal hearing was held before the undersigned on November 8, 2006, in Charleston, West Virginia, at which all parties were afforded full opportunity in accordance with the Rules of Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations,

Parts 410, 718, 725, and 727. At the hearing, I admitted Director's Exhibits (DX) 1 through 51, Employer's Exhibits (EX) 1 through 9, and Claimant's Exhibits (CX) 1 through 3.[1] The parties were provided with time to file post-hearing briefs. The Claimant filed her brief on February 26, 2007; the Employer filed its brief on February 13, 2007; the Director did not file a brief.

I have based my analysis on the entire record, including the exhibits, submitted briefs, and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

Jurisdiction and Procedural History

Mr. M. filed a claim for benefits on November 4, 2002, and was awarded benefits by the Director on November 21, 2003 (DX 2, 26). The Employer requested a hearing, and the file was transmitted to the Office of Administrative Law Judges on August 3, 2004 (DX 48).

Mr. M. filed a previous claim on June 5, 1973, which was denied by the Director on November 9, 1979 (DX 1). Mr. M. did not further pursue this claim.

The Claimant is Mr. M.'s wife; she filed a claim for benefits as his survivor on June 25, 2002. The Director issued a Proposed Decision and Order denying benefits on August 26, 2003. While the Director found that Mr. M. had contracted pneumoconiosis caused by his coal mine work, he concluded that the evidence was not sufficient to establish that pneumoconiosis contributed to his death (DX 12). The Claimant requested a hearing before the Office of Administrative Law Judges, and on January 7, 2004, the claim was forwarded to the Office of Administrative Law Judges (DX 14).

Issues

The issues contested by the Employer in connection with the living miner's claim are:

1. The timeliness of Mr. M.'s living miner's claim.
2. Whether the Claimant has established that Mr. M. had pneumoconiosis.
3. If so, whether Mr. M.'s pneumoconiosis was due to his coal mine employment.
4. Whether Mr. M. was totally disabled.
5. If so, whether Mr. M.'s totally disabling respiratory condition was due to pneumoconiosis.
6. Whether the evidence establishes a material change in condition.
7. Whether Mr. M.'s death was due to pneumoconiosis.

(Tr. 15-16).

Findings of Fact and Conclusions of Law

[1] The parties indicated that they were relying on the same medical evidence in both the living miner's and survivor's claims.

Background

The miner, Mr. B. D. M., was born on November 1, 1922; he obtained a GED (DX 2). In his application, Mr. M. claimed that he worked in the coal mines for Omar Mining Company for 46 years, retiring on February 2, 1988 (DX 2). Mr. M. married his wife, M. N., on July 1, 1950; they remained married until Mr. M.'s death on January 14, 2003 (DX 31). At the time of his application, Mr. M. did not have any dependent children. I find that Mr. M. had one dependent, his wife, for purposes of augmentation of benefits.

Mrs. M. testified at the hearing. She stated that her husband worked as a car dropper at the tipple. When he returned from work, he was covered with coal dust; his clothes were covered with coal dust. Mr. M. also worked as a mechanic and electrician at the coal mines. Mrs. M. testified that her husband had a hernia operation in the 1970s, and did not smoke after that. She thought that he started smoking in his teens; he smoked about a pack of cigarettes a day. According to Mrs. M., her husband coughed every day, and spit up black dust mixed with mucus.

The Director determined that Mr. M. had 40 years of coal mine employment, as documented by Mr. M.'s social security earnings records; the Employer does not contest this finding, or its designation as the responsible operator. Accordingly, I find that Mr. M. had at least 40 years of coal mine employment, and that the Employer is properly designated as the responsible operator.[2]

Living Miner's and Survivor's Claims

In connection with Mr. M.'s living miner's and Mrs. M.'s survivor's claim, the parties submitted the following evidence, in accordance with the limitations of the new guidelines.

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[2] The Employer also contests the timeliness of Mr. M.'s living miner's claim. The Claimant's original claim was filed on November 4, 2002. 20 C.F.R. § 725.308 provides that

> A claim for benefits filed under this part by, or on behalf of, a miner shall be filed within three years after a medical determination of total disability due to pneumoconiosis which has been communicated to the miner or a person responsible for the care of the miner, or within three years after the date of enactment of the Black Lung Benefits Act of 1977, whichever is later.

The regulations also provide that there is a rebuttable presumption that every claim for benefits is timely filed. 20 C.F.R. § 725.308(c). The Board has held that a determination of total disability due to pneumoconiosis must be "actually received" by the miner, and if so, there must be a finding that the miner was capable of understanding the report. *Adkins v. Donaldson Mine Co.,* 19 B.R.R 1-34 (1993).

There is nothing in the exhibit record to indicate that Mr. M. was diagnosed with a total disability due to pneumoconiosis at any time before he filed his application on November 4, 2002. Given the presumption that a claim is timely filed, and the total lack of any evidence to rebut this presumption, I find that Mr. M.'s claim for benefits was timely filed

X-ray Evidence[3]

Exhibit No.	Date of X-ray	Reading Date	Physician/ Qualifications	Impression
DX 25	12-11-02	8-19-03	Wheeler/B, BCR	Negative for pneumoconiosis
DX 17/16	12-11-02	2-28-03	Navani/B, BCR	Read for quality
DX 15	12-11-02	12-12-02	Hayes/B, BCR	1/0, q, q
EX 5	12-11-02	8-15-03	Scott/B, BCR	Negative for pneumoconiosis

Pulmonary Function Studies

Exhibit No.	Date	Age/Ht	FEV1	FVC	MVV	Effort	Qualifying[4]
DX 12	12-11-02	80/65.75"	0.60 0.64*	1.84 1.98*		Good	Yes Yes

* After administration of bronchodilators

Arterial Blood Gas Studies

Exhibit No.	Date	Physician	pCO2	pO2	At Rest/After Exercise
DX 11	12-11-02	Walker	57	43	At rest

Medical Opinions

[3] B-B reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

[4] A "qualifying" pulmonary function study yields values that are equal to or less than the appropriate values set out in the tables at 20 C.F.R. Part 718, Appendix B. A "non-qualifying" study exceeds those values. 20 C.F.R. §718.204(b)(2)(i).

Dr. James H. Walker

Dr. Walker examined Mr. M. at the Director's request on December 11, 2002 (DX 10). He reported Mr. M.'s occupational history, as well as his family and medical histories. On his examination of Mr. M., Dr. Walker noted that he was in poor general clinical condition. He was oxygen dependent, and short of breath at rest. Dr. Walker reported that Mr. M. had ankle edema; there was increased AP diameter of the chest, and palpation and percussion were normal. Breath sounds were quite suppressed, but there were marked rales and wheezing bilaterally; there was mild to moderate kyphosis.

Mr. M. told Dr. Walker that he had smoked a pack of cigarettes a day from the age of 15 to the age of 50. The x-ray administered by Dr. Walker showed pneumoconiosis 1/0, q, q, with bullous and generalized emphysema. Pulmonary function testing showed a severe obstructive ventilatory defect, with no significant response to bronchodilators. Arterial blood gas studies showed severe hypoxemia at rest. Dr. Walker's conclusion was that Mr. M. had coal workers' pneumoconiosis 1/0, q, q; bullous emphysema; chronic obstructive pulmonary disease; chronic bronchitis with bronchospasm; congestive heart failure by history, and vascular disease by history.

According to Dr. Walker, based on his severe obstructive ventilatory defect, hypoxemia, and positive x-ray findings, Mr. M. had an occupational lung disease caused by his coal mine employment. He rated Mr. M.'s pulmonary impairment as severe, and the result of his occupational dust exposure and tobacco abuse. He felt that Mr. M. did not have the respiratory capacity to perform his previous coal mining work, noting that he had a severe obstructive ventilatory defect, and that his blood gas testing showed severe hypoxemia at rest. Mr. M. was oxygen dependent, and his x-ray was positive for occupational pneumoconiosis.

Charleston Area Medical Center

The final discharge diagnosis from the Charleston Area Medical Center indicates diagnoses of acute respiratory failure, exacerbation of obstructive chronic bronchitis, congestive heart failure, hypertension, occlusion and stenosis of carotid artery, hypopotassemia, cerebral degeneration, cardiac dysrrhythmias, and history of tobacco abuse (DX 34).

Mr. M. was admitted on January 11, 2003 with exacerbation of COPD, shortness of breath, and a history of a fall. Dr. Loay Al-Asadi noted that Mr. M. had a longstanding history of advanced COPD. He also had a history of possible carotid stenosis, hypertension, and occasional congestive heart failure. Dr. Al-Asadi noted that he had diminished air entry bilaterally, with scattered end-expiratory wheezing. Dr. Al-Asadi's assessment included hypercapnic respiratory failure secondary to mild chronic obstructive pulmonary disease exacerbation, secondary to acute bronchitis; history of falling down, due to possible carotid stenosis, and possible small transient ischemic attack; history of cor pulmonale with mild right sided failure; mild hypokalemia; and rule out rib fracture.

Mr. M. was admitted on February 18, 2002, and discharged on February 25, 2002 with a diagnosis of decompensated COPD with purulent bronchitis, and cardiomyopathy. Dr. Juan D'Brot, who completed the discharge summary, noted that Mr. M. had a history of cardiomyopathy secondary to atherosclerotic heart disease, and chronic obstructive pulmonary disease related to smoking. Mr. M.'s physical examination showed that he was in moderate respiratory distress, with significant wheezes. His chest x-ray showed no acute infiltrate.

Dr. Al-Asadi saw Mr. M. on May 20, 2002, and noted that Mr. M.'s chest had fair entry bilaterally, with no active wheezing, rales, or rhonchi.

Dr. D'Brot saw Mr. M. on an office visit on February 13, 1997, after he was discharged from the hospital, where he was treated for severe hypoxemia and evidence of decompensated COPD with fluid retention. His echocardiogram showed evidence of pulmonary hypertension, and his physical examination showed bilateral wheezes. When Dr. D'Brot examined him on this visit, his lungs had fairly good air movement with occasional rhonchi, and no wheezes. Dr. D'Brot's diagnosis was chronic obstructive pulmonary disease associated with pulmonary hypertension and fluid retention, and peripheral vascular disease.

Office notes covering the period from April 1997 to October 2002 reflect that Mr. M. was treated for chronic obstructive pulmonary disease associated with pulmonary hypertension and water retention, diabetes mellitus, peripheral neuropathy, chronic hypoxia, obstructive sleep apnea, cardiomyopathy, alcoholism, atherosclerotic heart disease.

Mr. M. underwent a CT scan on February 24, 2002. Dr. Cordell reviewed the films, noting a small faint nodular density in the left mid lung laterally, which did not contain calcium.

Dr. Cordell reviewed Mr. M.'s February 13, 2002 x-ray, noting continued areas of increased density in the left lung base, consistent with atelectasis or scarring. He also noted chronic fibrotic changes in both lungs, with the interstitial markings in the right mid lung less prominent than on a previous examination. There continued to be blunting of the left costophrenic angle, unchanged from a previous examination, consistent with pleural adhesions or pleural fluid.

Dr. Reifsteck reviewed Mr. M.'s December 14, 2001 x-ray, noting the interval development of left basilar infiltrate or atelectasis, with a small left sided pleural effusion. There was no sign of new infiltrates, but there was old granulomatous disease and chronic interstitial changes.

Dr. Elksnis reviewed Mr. M.'s August 30, 2000 x-ray, noting scarring and emphysematous changes at the lung bases, and a calcified granuloma in the right lower lung zone. Dr. Elksnis also reviewed the August 22, 2000 x-ray, noting a questionable stellate density

in the left upper lung zone, likely due to superimposed bony vascular structures, and a calcified granuloma in the right lower lung zone.

Dr. Anton reviewed Mr. M.'s February 23, 2000 x-ray, reporting changes related to COPD, with bibasilar scarring. Dr. Anton also reviewed a CT scan performed the same day, noting a progression of emphysematous changes bilaterally, worse in the bases. There were linear areas of parenchymal scarring in the lung bases bilaterally, worse on the left, and a stable granuloma in the right lung base. He also described a 2 mm. nodular density in the superior segment of the left lower lung, which had not changed in size, consistent with a benign etiology.

Dr. P. M. Phillips read Mr. M's chest x-ray dated August 4, 1998, noting bilateral scarring at the bases, but no acute focal infiltrate or pleural effusions.

Mr. M. underwent cardiac testing on June 19, 1997. Dr. S. Willis Trammell, who wrote the report, stated that Mr. M. had clinically significant arterial occlusive disease in the right lower extremity, which appeared to be localized, and probably clinically significant arterial occlusive disease in the left lower extremity. An echocardiogram performed on January 8, 1997, showed that Mr. M. had normal LV systolic function, left pleural effusion, and moderately severe tricuspid regurgitation, with indirect evidence to suggest severe pulmonary hypertension, with almost systemic pulmonary artery pressures.

Pulmonary function testing performed on April 11, 1997, and on June 12, 1997, indicated severe obstruction, as well as low vital capacity, possibly from a concomitant restrictive defect.

Death Certificate

Dr. Al-Asidi completed Mr. M.'s death certificate, stating that the immediate cause of his death was chronic obstructive lung disease (DX 33). Dr. Al-Asidi did not have the autopsy results available before he completed this certificate.

Dr. Tomislav M. Jelic

Dr. Jelic performed the autopsy on Mr. M. (DX 35). He noted that Mr. M. had been admitted to the hospital with increased shortness of breath and productive cough; he had a longstanding history of chronic obstructive pulmonary disease, arterial hypertension, and congestive heart failure. His physical examination showed diminished air entry bilaterally with scattered end-expiratory wheezing, and mild bilateral pitting edema. Mr. M.'s x-ray showed granuloma in the right mid lung zone, otherwise clear.

On his gross examination of Mr. M.'s respiratory tract, Dr. Jelic noted that the major bronchial mucosa had a congested appearance with no gross evidence of aspiration. The right lung was hyper-expanded. The surface of the lungs was gray, and showed an anthracotic pattern. Dr. Jelic noted subpleural emphysematous bullae in the lower part of the upper and middle lobe.

The left lung was crepitant throughout, and cut sections of the parenchyma showed black dots. The right lung was subcrepitant, and cut sections showed dark red discoloration and black dots; in the upper part of the lower lobe there was one calcified nodule measuring 1.1 cm. in diameter.

On his microscopic examination, Dr. Jelic noted that sections of the lungs showed simple coal workers' pneumoconiosis, with acute bronchopneumonia in the right upper and lower lobes. There was organizing pneumonia in the middle, right lower, and left upper lobes. There was simple coal workers' pneumoconiosis, with multiple macules, throughout all lobes, and occasional dust nodules. Dr. Jelic found numerous anthracotic and polarizable silicate particles and conspicuous associated refractile collagen fibrosis. There was marked focal emphysema surrounding the macules. Dr. Jelic described acute exacerbation of chronic bronchitis, with focal squamous metaplasia of the respiratory epithelium. There was marked emphysema. The large anthracosilicotic nodule in the right lower lobe, for the most part, had ossification, with the presence of hematopoietic elements.

Dr. Jelic's diagnosis was simple coal workers' pneumoconiosis; marked emphysema with focal formation of bullae (bullous emphysema); acute exacerbation of chronic bronchitis; acute bronchopneumonia involving the right upper and lower lobes; organizing pneumonia involving the right middle and lower, and left upper lobes; adhesions between the left lung, chest wall, and diaphragm; and liver congestion.

Dr. Francis H. Y. Green

Dr. Green examined the autopsy slides at the Claimant's request, as well as Mr. M.'s medical records, and prepared a report dated June 13, 2006 (CX 1). On examination of the slides, he stated that the lungs appeared to be adequately sampled, and the sections were of good quality. There was simple coal workers' pneumoconiosis on all sections of the lung, predominantly of the macular type. There were also occasional micronodules, a single silicotic nodule, and occasional foci of interstitial fibrosis, which appeared to be dust related. Overall, the lesions of pneumoconiosis were moderately severe; they contained large quantities of black pigment, much of which had the morphology of bituminous coal mine dust, together with numerous birefringent needle-shaped particles, consistent with silicates, seen by polarizing microscopy. According to Dr. Green, all of the macules were characterized by coexistent focal (centriacinar) emphysema.

Dr. Green reported that overall, the emphysema was severe; the majority was of the focal/centriacinar type, but in some areas it had become the most severe form, panacinar. The large cartilaginous airways were not included in the sampling, and thus it was not possible to evaluate for chronic bronchitis. However, the smaller membranous bronchioles showed severe pathology, characterized by fibrosis of the walls, epithelial hyperplasia, and mucosal inflammation comprised of lymphocites and occasional eosinophils. The basement membrane was thickened, and there was mild mucous cell metaplasia. He stated that the changes in the membranous bronchioles appeared to have features both of chronic mineral dust exposure, and asthmatic type inflammation. But there was no evidence of current cigarette smoking.

According to Dr. Green, the pulmonary blood vessels showed marked abnormalities: the large elastic arteries showed atherosclerosis, the muscular pulmonary arteries were dilated and tortuous, and the arterioles had assumed a very thickened muscular media. He felt that these changes were consistent with severe pulmonary hypertension, which would correlate with clinical cor pulmonale. The lungs also showed changes of acute bronchitis, with early bronchopneumonia scattered throughout the lungs.

With respect to Mr. M.'s lungs, Dr. Green's diagnoses were moderately severe simple coal workers' pneumoconiosis, predominantly macular, but also showing micronodules, a silicotic nodule, and interstitial fibrosis; severe emphysema of the focal/centriacinar and panacinar types; severe pulmonary vascular changes consistent with pulmonary hypertension; membranous bronchiolitis with features of mineral dust and asthmatic etiologies; and early bronchopneumonia.

On considering Mr. M.'s medical records in conjunction with his review of the autopsy slides, Dr. Green concluded that there was little doubt that Mr. M. died from end-stage lung disease associated primarily with an obstructive impairment, and characterized at autopsy by the presence of pneumoconiosis, emphysema, and chronic bronchitis. According to Dr. Green, chronic bronchitis and emphysema are the pathologic hallmarks of the clinical diagnosis of COPD.

Dr. Green noted that he, Dr. Jelic, Dr. Oesterling, and Dr. Bush agreed that Mr. M. had evidence of simple pneumoconiosis, as well as evidence of severe emphysema. He noted that there were pulmonary vascular changes associated with severe pulmonary hypertension, which correlated well with the clinical history of cor pulmonale. As a terminal event, there was acute bronchitis and early bronchopneumonia. Dr. Green was not able to diagnose chronic bronchitis, as there was insufficient sampling of the large airways. However, there was a well-documented history of chronic cough and sputum. Mr. M.'s medium-sized airways showed changes of chronic inflammation with an asthmatic component, which would fit with the clinical history of a component of reversibility of the airway obstruction with bronchodilators.

According to Dr. Green, the chest x-rays taken during Mr. M.'s life tended to be read as negative for pneumoconiosis, or category 1 pneumoconiosis, but most of the readers documented calcified granulomata. Dr. Green stated that this diagnosis could not be made by radiologic means, and that calcified granulomata would have a similar radiographic appearance to silicotic nodules, which were found in the lungs at autopsy. He concluded that the radiologists under-read for the presence of pneumoconiosis. He also stated that the emphysema would contribute to the under-reading for pneumoconiosis.

There was no evidence of PMF, either clinically or at autopsy. In Dr. Green's opinion, the simple coal workers' pneumoconiosis found at autopsy was, in total, moderately severe, and comprised four elements: macules which predominated, micronodules, a silicotic nodule, and

interstitial fibrosis. All of these types of pneumoconiosis were caused by Mr. M.'s exposure to coal mine dust.

Dr. Green had no doubt, both clinically and pathologically, that Mr. M. had severe COPD, characterized by emphysema and chronic bronchitis. He acknowledged that cigarette smoking was a well-recognized cause of these conditions. But he stated that over the last thirty years, numerous studies have also shown that exposure to coal mine dust can cause emphysema and chronic bronchitis. He attached a list of studies indicating that one pack year of smoking contributes about the same degree to the decline in pulmonary function, or to the emphysema score, as does one year of exposure to coal mine dust. Mr. M. smoked about a pack a day for 35 years, quitting in 1987. He had a work history of 42 years in surface mines. In Dr. Green's opinion, both smoking and coal mine dust contributed about equally to Mr. M.'s COPD and pulmonary impairment. Dr. Green stated that studies have also shown that all types of emphysema are related to coal mine dust exposure, with the most common being centriacinar. Similarly, cigarette smoking is also primarily associated with centriacinar emphysema, but it is also related to the other types. Thus, it is not possible to differentiate dust-induced and cigarette-induced emphysema on the basis of its type.

In Dr. Green's opinion, all of Mr. M.'s medical pneumoconiosis was due to coal mine dust exposure, and about 50% of his COPD was due to coal mine dust exposure. Both would have contributed to his severe respiratory impairment, which had progressed to cor pulmonale, and which was the direct cause of his death. He felt that cigarette smoking would have contributed to Mr. M.'s COPD, and should also be considered a factor contributing to Mr. M.'s death. But the effects of Mr. M.'s smoking would be less than the effects of the coal dust, because smoking would not have contributed to the medical pneumoconiosis component. Thus, Dr. Green concluded that Mr. M. died of respiratory failure due to pneumoconiosis and COPD, and that coal mine dust exposure was the major underlying factor in his death, with cigarette smoking being a contributory factor.

Dr. Everett F. Oesterling

Dr. Oesterling examined the autopsy slides, reviewed Mr. M.'s medical records at the Employer's request, and prepared a report dated June 14, 2004 (EX 1). Dr. Oesterling reported his findings on a detailed examination of a slide showing the 2 mm. nodule in the left upper lobe of Mr. M.'s lung. Based on the structure, which included birefringent crystalline structures, he felt that there was evidence of a micronodular coalworkers' pneumoconiosis. He also examined a 7 mm. nodular structure, noting reticulin fibers that contained black pigment. He felt that the nodular structure appeared to be related to coal mine dust exposure, but the histologic pattern was more suggestive of a healed granuloma that could be encompassing mine dust in its periphery. He also noted that the location in the lower lobe would also suggest that this finding was not primarily of mine dust origin. Dr. Oesterling felt that it was safe to say that Mr. M. had moderate micronodular with macular coalworkers' pneumoconiosis. However, he felt that the level of this disease was insufficient to have significantly altered structure, and thus it would have produced little or no disability or respiratory impairment.

Dr. Oesterling stated that there were other changes in Mr. M.'s lung tissue. He described this disease process in detail, indicating that it was panlobular emphysema, which progressed to bullous emphysema, and was the primary etiology of Mr. M.'s chronic obstructive pulmonary disease. He also noted findings associated with exposure to cigarette smoke, but unrelated to mine dust exposure.

According to Dr. Oesterling, panlobular pulmonary emphysema is a disease process that is not attributable to mine dust exposure. He referred to a textbook indicating that mine dust exposure is specific to centriacinar emphysema, and unrelated to panacinar emphysema.

Dr. Oesterling noted that the macrophages on autopsy suggested that Mr. M. was still exposed to some component of tobacco smoke, either primarily or secondarily. He cited to studies showing that cigarette smoking is the most important cause of chronic bronchitis in the United States, and that it increases the risk of dying from chronic bronchitis. Thus, he felt that cigarette smoking should be considered as the "primary etiologic agent" in the evolution of Mr. M.'s chronic obstructive pulmonary disease.

According to Dr. Oesterling, the slides showed other significant pulmonary findings, including squamous metaplasia within the airways, which indicated that Mr. M.'s airways were exposed to an irritant, because squamous metaplasia is a reparative process seen with marked mucosal irritation. He felt that these findings indicated ongoing exposure to a noxious agent; as Mr. M. retired from coal mining in 1988, and his death occurred 15 years later, the airway irritation could not be attributed to an ongoing exposure to any substance within the mining environment. Dr. Oesterling concluded that a primary or secondary exposure to tobacco smoke was the more logical etiologic agent.

Dr. Oesterling also pointed to findings of fairly prominent passive congestion in the lung tissue, with superimposed bronchopneumonia; he stated that the passive congestion was associated with a failing left heart ventricle, and that the degree of change suggested fairly significant passive congestion. He noted that the prosector described pneumonia involving the right middle and lower lobes, and the left upper lobe, and thus the primary insult was passive congestion with superimposed pneumonia, which complicated Mr. M.'s already compromised pulmonary status due to panlobular and bullous emphysema. As further documentation of heart failure, Dr. Oesterling pointed to a slide from Mr. M.'s liver, which showed fairly prominent passive congestion, indicating heart failure.

Based on the histologic findings, Dr. Oesterling concluded that there was evidence of moderate micronodular with macular coalworkers' pneumoconiosis. But the changes due to mine dust appeared to be insufficient to have in any way contributed to Mr. M.'s death. Dr. Oesterling acknowledged that Mr. M. suffered from pulmonary and respiratory impairment before his death, due to panlobular and bullous emphysema, which was secondary to a significant smoking history. According to Dr. Oesterling, Mr. M. would have been partially disabled before his death, based on his emphysema and possible cardiac disease. But

coalworkers' pneumoconiosis and occupational dust exposure did not in any way contribute to any pulmonary or respiratory impairment suffered by Mr. M. Nor did it in any way hasten, contribute to, or cause Mr. M.'s death.

Dr. Oesterling testified by deposition on November 3, 2006 (EX 9). At the time he wrote his report, Dr. Oesterling did not have Dr. Green's report; he testified that it did not change any of his opinions. He noted that Dr. Green did not specifically describe the micronodular changes in the lung tissue, but talked much more about the macular disease. Dr. Green attributed the 1.1 cm area of nodular change described by the prosector to coal workers' pneumoconiosis; Dr. Oesterling thought that this was a healed calcified granuloma.

Dr. Oesterling also discussed his photographs of the panlobular emphysema in Mr. M.'s lungs. He pointed out the pattern and number of pulmonary macrophages, which are typically seen if there has been inhalation of cigarette smoke. He felt that Mr. M. had to be inhaling cigarette smoke, either actively or passively, at the time of his death, or there would not have been so many of the cells. He described the abnormal macrophages on his photographs as smokers' macrophages.

According to Dr. Oesterling, panlobular emphysema is typically seen with the inhalation of cigarette smoke, and in asthmatics. He felt that in Mr. M.'s case, there was one or both factors, cigarette smoke and asthmatic bronchitis.

Dr. Oesterling also discussed the squamous metaplasia in the small airways, which indicated that Mr. M. still had some form of irritation involving the airways; cigarette smoke is a common cause of such irritation. There was also inflammation underneath the areas of squamous metaplasia, what is typically seen in asthmatic bronchitis.

Dr. Oesterling discussed the passive congestion in Mr. M.'s lung tissue, which was an ideal medium for the growth of bacteria. He felt that Mr. M. had an ongoing problem with pneumonia, in addition to the acute bronchopneumonia. Passive congestion is typically associated with a failing left heart ventricle. Dr. Oesterling noted that Mr. M. had severe tricuspid regurgitation, which could have been one of the major factors in the passive congestion. He felt that Mr. M.'s pulmonary hypertension resulted from several things, including the leaking valve, as well as the failing left ventricle, and the emphysema.

Dr. Oesterling disagreed with Dr. Green's conclusion that it was not possible to differentiate between dust induced and cigarette induced emphysema based on its type. He felt that Mr. M. had pan-lobular emphysema, which is not related to coal mine dust, but is definitely seen in cigarette smokers, and asthmatics.

Dr. Oesterling also disagreed with Dr. Green's conclusion that about 50 percent of Mr. M.'s COPD was due to coal mine dust exposure. He did not see that much coal mine dust in the lungs, and there was no coal mine dust in the areas with emphysema. Thus, there was not a

significant enough dust burden to account for the extreme level of Mr. M.'s disease process. Nor did he think the literature supported such a conclusion.

Dr. George L. Zaldivar

Dr. Zaldivar reviewed Mr. M.'s medical records, and prepared a report dated March 21, 2005 (EX 2). He concluded that the records showed that Mr. M. had severe bullous emphysema, as found by CT scan, and by autopsy. He disagreed with Dr. Oesterling's assessment of possible left ventricular dysfunction, noting that the echocardiograms showed normal left ventricular function with severe pulmonary hypertension as of 1997. According to Dr. Zaldivar, it was not hard to appreciate the cause of the pulmonary hypertension, as Mr. M. was so hypoxic, and he had so much lung destruction. His was a case of cor pulmonale.

Dr. Zaldivar pointed to the autopsy report, which showed mild coal workers' pneumoconiosis, and severe bullous emphysema; there was bronchopneumonia terminally, and probably a cardiac arrhythmia resulting in pulmonary vascular congestion terminally. However, the terminal events were not documented. He felt that the records showed the typical abnormalities found in a smoker who developed bullous emphysema. As stated by Dr. Oesterling, bullous emphysema is not a manifestation of coal workers' pneumoconiosis, but is a manifestation of smoker's emphysema. He stated that the bullae were a magnification of the panacinar emphysema, or lung destruction, caused by smoking.

Dr. Zaldivar stated that the damage caused to the lungs by retained dust is directly related to the amount of dust retention, and he cited to several medical articles. He indicated that the authors found a relationship between centriacinar emphysema and coal workers' pneumoconiosis, but not between panacinar or bullous emphysema and coal workers' pneumoconiosis.

In Dr. Zaldivar's opinion, Mr. M.'s pulmonary hypertension was a result of chronic hypoxemia and hypercarbia, as noted in the blood gases and the severe airway obstruction brought about by destruction of lung tissue, manifested by x-ray and pathologically as bullous emphysema. But this pulmonary hypertension was absolutely unrelated to the small amount of dust that was found in Mr. M.'s lungs on x-ray and at autopsy.

Dr. Zaldivar quoted a study by Kinsella, in a textbook entitled High Resolution CT of the Lung, published in 2001:

> The extent of emphysema was the strongest independent predictor of pulmonary function impairment; the extent of small nodules was also an independent predictor of pulmonary function impairment, albeit a weaker one. It was also shown in this study that in the absence of progressive massive fibrosis, smokers had more extensive emphysema and more severe function impairment than did nonsmokers. In the absence of progressive massive fibrosis, silicosis was not associated with significant emphysema.

Thus, according to Dr. Zaldivar, "once again" we see the association of coal workers' pneumoconiosis and silicosis with centriacinar emphysema or focal emphysema, but not with bullous or panacinar emphysema.

Dr. Zaldivar concluded that there was evidence to justify a diagnosis of coal workers' pneumoconiosis, and pulmonary impairment. This impairment was the result of bullous emphysema caused by smoking, and unrelated to Mr. M.'s occupation. From a pulmonary standpoint, Mr. M. was severely and permanently disabled from performing his usual coal mining job. But this pulmonary impairment was strictly due to his past smoking habit, and unrelated to his coal mine occupation. As a whole man, Mr. M. had a reduced capacity to perform his usual coal mining job, due to a combination of severe bullous emphysema and cardiac disease. But his past coal dust exposure did not have any role in his disability. And neither the pneumoconiosis found at autopsy nor Mr. M.'s coal dust exposure played any role in his death, nor did it hasten his death. In Dr. Zaldivar's opinion, Mr. M. would have died when and as he did even if he never worked in the coal mines.

Dr. Zaldivar testified by deposition on August 29, 2005 (EX 6). He testified that it "made sense" that Mr. M.'s death certificate stated that the immediate cause of his death was chronic obstructive lung disease. He stated that a death certificate is a reflection of previous admissions and treatment, and according to the treatment notes from Mr. M.'s doctors, he had had chronic obstructive disease and pulmonary hypertension for a while. He also had problems with peripheral neuropathy and diabetes. But his doctors were treating him primarily for chronic obstructive lung disease. Mr. M. was very hypoxic as a result of this condition, so it was understandable that his death certificate reflected that diagnosis.

Dr. Zaldivar described bullous emphysema as an empty space in the lungs of at least one centimeter in diameter with no blood vessels, and which does not participate in gas exchange; it has nothing to do with coal workers' pneumoconiosis, but is due to smoking. Coal dust exposure results in macules, with associated focal emphysema; it cannot cause bullous emphysema, without the macule and focal emphysema.

Dr. Zaldivar explained that Dr. Wiot reviewed a standard CT scan, and he did not see anything; a high resolution CT scan may have shown something. He cited to a study where micro-nodules were seen on high resolution CT scan, but not on chest x-ray.

Dr. Zaldivar also discussed a study showing that the amount of centriacinar emphysema and focal emphysema in coal miners was directly related to the amount of dust retained, as seen by pathologists. He stated:

> So the dust is causing the damage. And if the dust isn't there, then there couldn't be any damage resulting from the dust. That is the logic behind technically having pneumoconiosis, but physiologically having no consequences from it.

In Mr. M.'s case, he did not feel that part of his pulmonary impairment was the result of, or related to, his coal dust exposure, because any damage produced by such an insignificant

amount of dust, which was not seen by CT scan, but was seen only by the pathologist, could not be expected to produce any damage to the lungs. Again, he noted that the pathologist found bullous emphysema, which was what was causing the damage. Bullous emphysema was also found clinically. But there was no contribution from the small nodules to the overall damage to the lungs.

Dr. Zaldivar concluded that Mr. M. had cor pulmonale; his left ventricle was working well, but his right ventricle was not, and he had high pulmonary artery pressure. He stated that Mr. M.'s cor pulmonale could have been eliminated by continuous oxygen use; he was not sure that Mr. M. was on continuous oxygen.

Dr. Zaldivar testified that in his opinion, Mr. M. had bullous emphysema, which caused pulmonary problems unrelated to his work in the coal mines. His death was caused by severely advanced emphysema and some degree of heart disease, in addition to pulmonary hypertension. But neither coal dust exposure nor coal workers' pneumoconiosis caused or hastened his death.

Dr. James R. Castle

Dr. Castle reviewed Mr. M.'s medical records at the Employer's request, and prepared a report dated May 31, 2005 (EX 3). He concluded that Mr. M. had pathologic evidence of simple coal workers' pneumoconiosis. Dr. Castle noted that Mr. M. worked in the underground coal mining industry for a sufficient time to develop pneumoconiosis if he were a susceptible host. But another risk factor for the development of pulmonary disease is tobacco abuse. Mr. M. had at least a 35 pack year smoking history, sufficient to cause him to develop chronic obstructive pulmonary disease or lung cancer or atherosclerotic cardiovascular disease if he were a susceptible host. According to Dr. Castle, another risk factor for the development of pulmonary symptoms is cardiac disease. Mr. M. had a history of cardiomyopathy, which can result in significant shortness of breath.

Dr. Castle stated that at no time did Mr. M. demonstrate consistent physical findings indicating the presence of an interstitial pulmonary process; he did not have consistent findings of rales, crackles, or crepitations. He noted that the majority of the x-ray readers did not find pneumoconiosis radiographically, but Mr. M. did have bullous emphysema, primarily in the basilar or lower lung zones. His physiologic studies showed evidence of severe airway obstruction, and arterial blood gas studies showed a severe degree of hypoxemia associated with hypercapnia.

Dr. Castle pointed out that all of the pathologists found evidence of simple coal workers' pneumoconiosis; Dr. Bush and Dr. Oesterling felt that it was of insufficient severity to have caused any disability, or to have played any role in Mr. M.'s death.

Dr. Castle concluded that Mr. M. had a severe respiratory impairment caused by his smoking habit. His death was not caused by, contributed to, or hastened by coal workers' pneumoconiosis. Rather, his death was due to tobacco smoke induced bullous emphysema, complicated by acute bronchopneumonia. He would have died when and as he did, regardless of his occupational history or the presence of pneumoconiosis.

Dr. Castle testified by deposition on August 31, 2005 (EX 7). He discussed Mr. M.'s final hospital admission, when he had respiratory failure related to his bullous emphysema; he did not improve, but deteriorated and died. His autopsy showed that he had an acute bronchopneumonia involving the upper and lower right lobes, and some organizing pneumonia in the right middle and lower, and left lobes, meaning that he had pneumonia for a number of days before his death. He felt that Mr. M.'s death was from pneumonia that complicated his bullous emphysema.

Dr. Castle testified that bullous emphysema is not caused by simple pneumoconiosis, nor is it related to coal mine dust. Coal mine dust causes focal emphysema as part of the coal macule, and that was seen, but Mr. M. also had large areas of bullous emphysema unrelated to the coal macules or coal mine dust. Mr. M. also had a history of congestive heart failure, and cardiomyopathy.

Again, Dr. Castle stated that Mr. M. had pathologic evidence of simple pneumoconiosis. He also had a severe degree of bullous emphysema as a result of his smoking habit. His pneumoconiosis did not play any role in his respiratory impairment. Mr. M. died as a result of acute and subacute pneumonia complicating his bullous emphysema. But neither his pneumoconiosis nor his exposure to coal dust played any role in his death.

Dr. Jerome F. Wiot

Dr. Wiot reviewed CT scans performed on February 23, 2000 and February 24, 2002 at the Employer's request (EX 4). He stated that the February 23, 2000 CT scan was done with mediastinal windows only, and thus could not be evaluated for the presence or absence of pneumoconiosis. With respect to the February 24, 2002 CT scan, Dr. Wiot stated that it showed no evidence of coal workers' pneumoconiosis. He noted that the lung fields were somewhat over-expanded, consistent with emphysema. There was prominent atherosclerotic change in the thoracic aorta, and a few linear strands at the left base, undoubtedly due to disc atelectasis related to Mr. M.'s poor inspiration. He noted a pleuropericardial adhesion on the right, which was not a manifestation of coal dust exposure, and a few scattered calcified granulomas.

According to Dr. Wiot, CT scans are medically acceptable for the evaluation of pulmonary problems. They are beneficial in confirming or denying the presence of simple pneumoconiosis, and can be beneficial in recognizing complicated pneumoconiosis when it is not evident on the routine x-rays.

DISCUSSION

Change in Condition of Entitlement

Mr. M.'s living miner's claim is a "duplicative" or "subsequent" claim because a previous claim was finally denied over one year ago. There is, accordingly, a threshold issue as to whether there are grounds for reopening the claim under 20 C.F.R. §725.309. A subsequent claim will be denied unless the claimant can demonstrate that at least one of the conditions of

entitlement upon which the prior claim was denied ("applicable condition of entitlement") has changed and is now present.[5] 20 C.F.R. §§725.309(d)(2), (3). If a claimant does demonstrate a change in one of the applicable conditions of entitlement, then generally findings made in the prior claim(s) are not binding on the parties. 20 C.F.R. §725.309(d)(4). Consequently, the relevant inquiry in a subsequent claim is whether evidence developed since the prior adjudication would now support a finding of a previously denied condition of entitlement.

Mr. M.'s previous living miner's claim was finally denied because he did not establish the existence of pneumoconiosis or total disability due to pneumoconiosis. (DX 1) Thus, for purposes of adjudicating the "subsequent" claim, I must first evaluate whether Mrs. M. has established either of these elements of entitlement by a preponderance of the newly submitted evidence.

Existence of Pneumoconiosis[6]

Section § 718.202 provides four means by which pneumoconiosis may be established. Under § 718.202(a)(1), a finding of pneumoconiosis may be made on the basis of the x-ray evidence. The record includes three readings of an x-ray performed on December 11, 2002.[7] Dr. Hayes, who is dually qualified, read this x-ray as positive for pneumoconiosis, category 1/0. However, Dr. Wheeler and Dr. Scott, who are also dually qualified, read this x-ray as negative for pneumoconiosis. As the preponderance of readings by the most qualified physicians is negative, I find that Mrs. M. has not established that Mr. M. had pneumoconiosis by the x-ray evidence.

Under § 718.202(a)(2), a finding of pneumoconiosis may be made on the basis of biopsy or autopsy evidence. There is no biopsy evidence in this case. However, Dr. Jelic performed a limited autopsy at Mrs. M.'s request. On his gross examination of Mr. M.'s lungs, Dr. Jelic noted that the surface showed an anthracotic pattern. There were also subpleural emphysematous bullae in the upper and middle lobe. Cut sections from both lungs showed black dots, and there was a calcified nodule in the upper part of the lower right lobe. On microscopic examination, Dr. Jelic found that sections showed simple pneumoconiosis, and acute bronchopneumonia in the right upper and lower lobes. He found simple pneumoconiosis, with multiple macules throughout all lobes, and occasional dust nodules. Dr. Jelic noted numerous anthracotic and polarizable silicate particles, and conspicuous associated refractile collagen fibrosis; there was marked focal emphysema surrounding the macules. According to Dr. Jelic, there was acute exacerbation of chronic bronchitis, and marked emphysema; the large anthracosilicotic nodule in the right lower lobe had ossification. His diagnosis was simple pneumoconiosis, marked emphysema with focal formation of bullae, acute exacerbation of

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[5] For a miner, the conditions of entitlement include whether the individual (1) is a miner as defined in this section; (2) has met the requirements for entitlement to benefits by establishing pneumoconiosis, its causal relationship to coal mine employment, total disability, and contribution by the pneumoconiosis to the total disability; and (3) has filed a claim for benefits in accordance with this part. 20 C.F.R. §725.202(d) (*Conditions of entitlement: miner*).

[6] As the parties have designated the same evidence in connection with both the living miner's and survivor's claim, my finding on this issue will apply to both claims.

[7] Dr. Navani read this x-ray for quality purposes only.

chronic bronchitis, acute bronchopneumonia involving the right upper and lower lobes, organizing pneumonia involving the right middle and lower, and left upper lobes; adhesions between the left lung, chest wall, and diaphragm; and liver congestion.

Dr. Oesterling examined the autopsy slides at the Employer's request. Based on his examination of a slide that showed a 2 mm. nodule in the left upper lobe, which included birefringent crystalline structures, he concluded that there was evidence of a micronodular coalworkers' pneumoconiosis. After his review of the slides, Dr. Oesterling felt that it was safe to say that Mr. M. had moderate micronodular with macular coalworkers' pneumoconiosis.

As both Dr. Jelic and Dr. Oesterling agree that the autopsy tissue slides show pneumoconiosis, I find that Mrs. M. has established that Mr. M. had pneumoconiosis by virtue of the autopsy evidence.

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found applicable. In the instant case, the presumption of § 718.305 does not apply to claims filed after January 1, 1982. Section 718.306 does not apply to claims where the miner died after March 1, 1978. Section 718.304 allows a presumption of complicated pneumoconiosis where, *inter alia*, an x-ray "yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C" if such miner is suffering or suffered from a chronic dust disease of the lung. 20 C.F.R. § 718.304(a). As there are no such x-ray findings in this case, this presumption does not apply.

Under § 718.202(a)(4), Mrs. M. can also establish that Mr. M. suffered from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See,* 20 C.F.R. § 718.107, *Hoffman v. B&G Construction Co.,* 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.,* 7 B.L.R. 1-295 (1984). A report which is better supported by the objective medical evidence of record may be accorded greater probative value. *Minnich v. Pagnotti Enterprises, Inc.,* 9 B.L.R. 1-89, 1-90 n.1 (1986); *Wetzel v. Director, OWCP,*8 B.L.R. 1-139 (1985).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra.* Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder of fact to decide. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned medical reports which support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP,* 11 B.L.R. 1-135 (1988). An equivocal opinion, however, may be given little weight. *Justice v. Island Creek Coal Co.,* 11 B.L.R. 1-91 (1988); *Snorton v. Zeigler Coal Co.,* 9 B.L.R. 1-106 (1986).

Dr. Walker, who examined Mr. M. at the Director's request, concluded that Mr. M. had pneumoconiosis, based on his positive x-ray findings. Although Dr. Walker's conclusion is consistent with the later autopsy findings, I have found that the x-ray evidence does not establish the existence of pneumoconiosis, thus calling into question the basis for Dr. Walker's conclusions. Accordingly, I have not accorded significant weight to Dr. Walker's determination regarding the existence of pneumoconiosis.[8]

Dr. Zaldivar, who reviewed Mr. M.'s medical records at the Employer's request, noting that the autopsy report showed mild coal workers' pneumoconiosis. He concluded that there was evidence to justify a diagnosis of coal workers' pneumoconiosis. Similarly, Dr. Castle, who reviewed Mr. M.'s medical records at the Employer's request, noted that all of the pathologists found evidence of simple pneumoconiosis.

Mr. M.'s treatment and hospitalization records show that he was treated for COPD and cardiomyopathy, and that he had a long history of advanced COPD. He also had a history of pulmonary hypertension and fluid retention, and cor pulmonale with mild right sided failure. But his medical records do not include any evaluation for or diagnosis of pneumoconiosis, or any coal dust related disease.

Based on the reports by Dr. Zaldivar and Dr. Castle, who relied on the autopsy findings, I find that Mrs. M. has established that Mr. M. had pneumoconiosis by a preponderance of the medical opinion evidence.

Finally, I have weighed all of the evidence under § 718.202(a) together, including the x-ray evidence, and I find that Mrs. M. has met her burden to establish that Mr. M. had pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 2000 WL 524798 (4th Cir. 2000).

Because Mrs. M. has established that Mr. M. had pneumoconiosis, she has established a material change in Mr. M.'s condition, and she is entitled to a consideration of Mr. M.'s claim on the merits.

Merits of the Living Miner's Claim

The record in connection with Mr. M.'s previous claim refers to an x-ray performed sometime before March 18, 1972; Dr. W. K. C. Morgan reported that it did not show pneumoconiosis (DX 1). However, I note that pneumoconiosis is a progressive disease, and the autopsy findings after Mr. M.'s death established that he had pneumoconiosis. Accordingly, I find that Mrs. M. has established that Mr. M. had pneumoconiosis.

[8] Dr. Walker's report suggests that he attributed Mr. M.'s severe obstructive disease in part to his occupational coal dust exposure. However, he did not explain the basis for such a conclusion.

However, Mrs. M. must also establish that Mr. M. was totally disabled by pneumoconiosis. The regulations as amended provide that a claimant can establish total disability by showing pneumoconiosis prevented the miner "[f]rom performing his or her usual coal mine work," and "[f]rom engaging in gainful employment in the immediate area of his or her residence requiring the skills or abilities comparable to those of any employment in a mine or mines in which he or she previously engaged with some regularity over a substantial period of time." 20 C.F.R. § 718.204(b)(1).

Total disability may be established by pulmonary function tests, arterial blood gas tests, evidence of cor pulmonale with right-sided congestive heart failure, or physicians' reasoned medical opinions, based on medically acceptable clinical and laboratory diagnostic techniques, to the effect that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in the miner's previous coal mine employment. 20 C.F.R. § 718.204(b)(2). Furthermore, under 20 C.F.R. § 718.304, if a claimant can establish the existence of "complicated pneumoconiosis," an irrebuttable presumption arises that the claimant is totally disabled due to pneumoconiosis. For a living miner's claim, total disability may not be established solely by the miner's testimony or statements. 20 C.F.R. § 718.204(d)(5).

The pulmonary function and arterial blood gas studies performed by Dr. Walker produced values that qualify for a presumption of total respiratory disability.[9] This presumption has not been rebutted: Dr. Walker concluded that Mr. M. had a severe and disabling pulmonary impairment. Although Dr. Jelic, Dr. Green, and Dr. Oesterling did not directly address the question of whether Mr. M. had a disabling pulmonary impairment, their reports contain findings of severe emphysema and obstructive disease. Dr. Zaldivar and Dr. Castle also concluded that Mr. M. had a severe and disabling pulmonary impairment. Finally, Mr. M.'s treatment records confirm that he had severe obstructive disease, with exacerbations that required hospitalization. Accordingly, I find that Mrs. M. has established that Mr. M. had a respiratory impairment that prevented him from performing his previous coal mining work.[10]

Finally, Mrs. M. must establish that Mr. M.'s disabling respiratory impairment was caused by pneumoconiosis. In this regard, Dr. Walker concluded that Mr. M.'s severe pulmonary impairment was the result of a combination of his occupational dust exposure and tobacco abuse. But Dr. Walker did not discuss the reasoning behind his conclusion, or offer any rationale or support for this diagnosis.

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[9] The regulatory table lists values only up to age 71. However, the qualifying values decrease consistently with age; as Mr. M.'s values would qualify for a man of 71, it is clear that, following the progression of these values downward, they would also qualify for a man of 80.

[10] There is also evidence that Mr. M. suffered from cor pulmonale: Mr. M.'s medical records document repeated clinical findings of cor pulmonale, with right sided heart failure. Dr. Green reported that Mr. M.'s pulmonary blood vessels showed marked abnormalities, consistent with severe pulmonary hypertension, which would correlate with clinical cor pulmonale. Dr. Zaldivar also acknowledged that Mr. M. had cor pulmonale, with right sided problems. I find that Mrs. M. has also established that Mr. M. had a totally disabling respiratory impairment in the form of cor pulmonale with right sided heart failure.

Dr. Green concluded that Mr. M. had severe COPD, characterized by emphysema and chronic bronchitis, both clinically and pathologically. Citing to studies showing that exposure to coal mine dust can cause emphysema and chronic bronchitis, he concluded that about half of Mr. M.'s COPD, which contributed to his severe respiratory impairment, was due to his coal mine dust exposure, with cigarette smoking also contributing to that impairment.

On the other hand, Dr. Oesterling felt that the level of the pneumoconiosis found on autopsy was not sufficient to have significantly altered structure, and would have produced little or no disability or respiratory impairment. Dr. Oesterling felt that the primary etiology of Mr. M.'s chronic obstructive pulmonary disease was his emphysema, which was not attributable to his coal dust exposure. Dr. Oesterling cited to a textbook that indicated that coal mine dust exposure is specific to centriacinar emphysema, and unrelated to panacinar emphysema. Because the autopsy slides suggested that Mr. M. was still exposed to tobacco smoke, either primarily or secondarily, he concluded that cigarette smoking should be considered as the "primary etiologic agent" in the evolution of Mr. M.'s chronic obstructive pulmonary disease.

Dr. Oesterling specifically stated that coal mine dust exposure is specific to centriacinar emphysema. But he did not address Dr. Green's conclusion that the majority of Mr. M's severe emphysema was focal/centriacinar, in some areas having progressed to the most severe form, panacinar, or the studies cited by Dr. Green showing that exposure to coal mine dust can cause emphysema and chronic bronchitis, with smoking contributing about the same degree to the decline in pulmonary function as one year of exposure to coal mine dust. Dr. Oesterling did not explain why he completely excluded Mr. M.'s 40 years of coal dust exposure as a factor in his development of obstructive disease, and resulting respiratory impairment. Rather, he focused on the pattern and number of pulmonary macrophages, which he stated were typically seen with the inhalation of cigarette smoke, and concluded that Mr. M.'s emphysema was due solely to cigarette smoke, and possibly asthmatic bronchitis.[11]

Dr. Zaldivar concluded that Mr. M. had bullous emphysema, a magnification of panacinar emphysema, which was caused by smoking. He cited to medical articles that found a relationship between centriacinar emphysema and pneumoconiosis, but not between panacinar emphysema and pneumoconiosis. He felt that the insignificant amount of dust seen by the pathologist could not be expected to produce any damage to Mr. M.'s lungs, and thus he felt that his pulmonary impairment was not related to his coal dust exposure. He noted that bullous emphysema was found by the pathologist. But although he acknowledged the relationship between coal mine dust exposure and focal/centriacinar emphysema, Dr. Zaldivar did not address Dr. Jelic's microscopic findings of marked focal emphysema surrounding the multiple macules of pneumoconiosis that were throughout all of the lobes. Nor did he discuss Dr. Green's microscopic findings lf macular lesions in all lung sections with numerous birefringent particles, all characterized by coexistent focal emphysema.[12]

[11] Dr. Oesterling did not explain why, even if cigarette smoke was the "primary" etiologic factor in the evolution of Mr. M.'s obstructive lung disease, his significant history of exposure to coal mine dust could not have been a coexistent factor, even if not the "primary" factor.

[12] Indeed, Dr. Green concluded that most of the emphysema he saw was focal, with some areas having the more severe panacinar emphysema.

Dr. Castle concluded that Mr. M.'s severe respiratory impairment was caused by his cigarette smoking, and that pneumoconiosis did not play any role in this respiratory impairment. He stated that bullous emphysema is not caused by pneumoconiosis or related to exposure to coal mine dust. However, he acknowledged that focal emphysema was seen, and that coal mine dust causes focal emphysema.

Both Dr. Green and Dr. Oesterling agreed that Mr. M.'s emphysema had progressed to the more severe panacinar, or bullous type. However, Dr. Oesterling ignored the findings by Dr. Dr. Jelic of marked focal emphysema surrounding multiple macules of pneumoconiosis throughout all lobes. While Dr. Castle agreed that there was focal emphysema seen on autopsy, neither he nor Dr. Zaldivar even considered the possibility that the focal emphysema (described by Dr. Green/Jelic as) seen in Mr. M.'s lungs on autopsy could be the result of Mr. M.'s lengthy exposure to coal mine dust, or could be a factor in his total respiratory impairment.

Dr. Castle acknowledged that there was focal emphysema, and that coal mine dust causes focal emphysema as part of the coal macule, but he did not explain how, in light of these findings, he was able to totally exclude Mr. M.'s 40 year history of coal dust exposure as a factor in his disabling emphysema.

For these reasons, I find that the opinions by Dr. Oesterling, Dr. Zaldivar, and Dr. Castle are unpersuasive, as they ignore critical pathologic findings by both Dr. Jelic and Dr. Green. I rely on Dr. Green's opinion, which is consistent with the autopsy findings by Dr. Jelic, and which discusses and addresses both the focal and panacinar emphysema found in Mr. M.'s lungs, and takes into account both Mr. M.'s extensive history of coal mine employment, as well as his lengthy, but also distant, history of cigarette smoking. I find that Mrs. M. has established by a preponderance of the medical evidence that Mr. M. was totally disabled due to pneumoconiosis. Accordingly, she is entitled to benefits under the Act in connection with the living miner's claim.

Survivor's Claim

To be entitled to benefits, Mrs. M. must establish that her husband's death was due to pneumoconiosis. Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c), as amended, which states, in pertinent part:

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at § 718.304 is applicable.

(4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.

(5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

Dr. Green concluded that the direct cause of Mr. M.'s death was his severe respiratory impairment, which had progressed to cor pulmonale. He felt that both Mr. M.'s medical pneumoconiosis, as well as his COPD, contributed to that respiratory impairment, and that Mr. M.'s medical pneumoconiosis was due to his coal mine dust exposure, and his COPD was due to a combination of his coal mine dust exposure and cigarette smoking. Dr. Green felt that the effects of cigarette smoking were less than the effects of coal dust exposure, as cigarette smoking would not have contributed to the medical pneumoconiosis component. Thus, coal mine dust was the major underlying factor in Mr. M.'s death, with cigarette smoking being a contributory factor.

Dr. Oesterling felt that the degree of pneumoconiosis found on autopsy was not sufficient to have contributed to Mr. M.'s death. Although he discussed the etiology of Mr. M.'s pulmonary impairment at length, Dr. Oesterling did not offer an opinion on the cause of Mr. M.'s death.

Dr. Zaldivar concluded that neither the pneumoconiosis found at autopsy nor Mr. M.'s coal dust exposure played any role in his death. He felt that it made sense that Mr. M.'s death certificate stated the immediate cause of his death was chronic obstructive lung disease. Dr. Zaldivar concluded that Mr. M. had bullous emphysema, and that his death was caused by severely advanced emphysema and some degree of heart disease, as well as pulmonary hypertension.

Although he concluded that Mr. M. had a severe respiratory impairment caused by his cigarette smoking, Dr. Castle stated that Mr. M.'s death was not caused by or hastened by pneumoconiosis. He felt that Mr. M.'s death was due to pneumonia that complicated his bullous emphysema.

But again, neither Dr. Oesterling, Dr. Zaldivar, nor Dr. Castle discussed the role that the marked focal emphysema surrounding the macules of pneumoconiosis throughout the lungs, as found by Dr. Jelic on autopsy, or the predominantly macular pneumoconiosis in all lung sections as found by Dr. Green, had in the development of Mr. M.'s disabling obstructive impairment, which none dispute led to his death. Dr. Oesterling seized on bullous emphysema as the global explanation for Mr. M.'s impairment, without explanation excluding focal emphysema, which he agreed was caused by coal dust exposure, as a factor. Dr. Zaldivar agreed that there was a relationship between exposure to coal mine dust and the development of focal emphysema, but

did not explain how he concluded that coal mine dust exposure played no role in Mr. M.'s death, in light of the findings of focal emphysema surrounding macules of pneumoconiosis in all sections of Mr. M.'s lungs. Dr. Castle acknowledged the findings of focal emphysema, which he agreed is caused by exposure to coal mine dust, but he did not explain its role in Mr. M.'s impairment or death, in particular, whether Mr. M.'s focal emphysema hastened or contributed to his acknowledged respiratory death.

I rely on the opinions of Dr. Green, who discussed all of the available medical information, and took into consideration both Mr. M.'s long history of coal mine employment, as well as his exposure to cigarette smoke, and thoroughly discussed all of the findings on autopsy. Dr. Green concluded that Mr. M. had pneumoconiosis in four forma; in macules, micronodules, a silicotic nodule, and interstitial fibrosis, all due to his exposure to coal mine dust. In addition, he had chronic obstructive pulmonary disease, due primarily to his exposure to coal mine dust, but also to his history of cigarette smoking. Both Mr. M's medical and legal pneumoconiosis contributed to his severe respiratory impairment, which progressed to cor pulmonale, and was a direct cause of his death. Relying on Dr. Green's opinions, I find that Mrs. M. has established that Mr. M.'s death was due to pneumoconiosis.

CONCLUSION

I find that Mrs. M. has established that Mr. M. had pneumoconiosis that arose from his coal mine employment.[13] In addition, she has established that Mr. M. was totally disabled due to pneumoconiosis, and that his death was due to pneumoconiosis. Accordingly, she is entitled to benefits under the Act in connection with the living miner's and survivor's claims.

ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claims of M. M., surviving spouse of B. D. M., for black lung benefits under the Act are GRANTED.

IT IS FURTHER ORDERED that the Employer, Omar Mining Company, shall pay to the Claimant all benefits to which she is entitled under the Act in connection with her survivor's claim.

IT IS FURTHER ORDERED that the Employer, Omar Mining Company, shall pay to the Claimant all benefits to which she is entitled under the Act in connection with the living miner's claim commencing in November 2002. [14]

[13] As Mr. M. worked for 40 years as a coal miner, he is entitled to the presumption, which has not been rebutted, that his pneumoconiosis arose from his coal mine employment.

[14] As the record does not establish precisely when Mr. M. became totally disabled due to pneumoconiosis, I have used his date of application as the date of onset of benefits.

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LINDA S. CHAPMAN
Administrative Law Judge

ATTORNEYS FEES

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).